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14. ABSTRACT <p>This investigation sought to study changes in leukocyte subsets after an acute bout of resistance exercise (ARE) and to determine whether ingestion of carbohydrate (CHO) could attenuate those immune responses. Nine male track-and-field athletes (21.1 ± 1.4 yr, 177.2 ± 5.5 cm, 80.9 ± 9.7 kg, $8.7\% \pm 3.8\%$ fat) and 10 male ice hockey athletes (21.0 ± 2.2 yr, 174.3 ± 6.2 cm, 79.6 ± 11.1 kg, $13.9\% \pm 3.73\%$ fat) participated in 2 different ARE protocols. Both experiments employed a counterbalanced double-blind research design, wherein participants consumed either a CHO (1 g/kg body weight) or placebo beverage before, during, and after a weight-lifting session. Serum cortisol decreased ($p < .05$) at 90 min into recovery compared with immediately postexercise. Plasma lactate, total leukocyte, neutrophil, and monocyte concentrations increased ($p < .05$) from baseline to immediately postexercise. Lymphocytes decreased significantly ($p < .05$) from baseline to 90 min postexercise. Lymphocytes were lower ($p < .05$) for the CHO condition than for placebo. The findings of this study indicate the following: ARE appears to evoke changes in immune cells similar to those previously reported during endurance exercise, and CHO ingestion attenuates lymphocytosis after ARE.</p>					
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Carbohydrate Supplementation and Immune Responses After Acute Exhaustive Resistance Exercise

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This investigation sought to study changes in leukocyte subsets after an acute bout of resistance exercise (ARE) and to determine whether ingestion of carbohydrate (CHO) could attenuate those immune responses. Nine male track-and-field athletes (21.1 ± 1.4 yr, 177.2 ± 5.5 cm, 80.9 ± 9.7 kg, $8.7\% \pm 3.8\%$ fat) and 10 male ice hockey athletes (21.0 ± 2.2 yr, 174.3 ± 6.2 cm, 79.6 ± 11.1 kg, $13.9\% \pm 3.73\%$ fat) participated in 2 different ARE protocols. Both experiments employed a counterbalanced double-blind research design, wherein participants consumed either a CHO (1 g/kg body weight) or placebo beverage before, during, and after a weight-lifting session. Serum cortisol decreased ($p < .05$) at 90 min into recovery compared with immediately postexercise. Plasma lactate, total leukocyte, neutrophil, and monocyte concentrations increased ($p < .05$) from baseline to immediately postexercise. Lymphocytes decreased significantly ($p < .05$) from baseline to 90 min postexercise. Lymphocytes were lower ($p < .05$) for the CHO condition than for placebo. The findings of this study indicate the following: ARE appears to evoke changes in immune cells similar to those previously reported during endurance exercise, and CHO ingestion attenuates lymphocytosis after ARE.

Keywords: cortisol, immune function, weight training

The impact of exercise on the immune system is important because athletic performance and ability to train might be compromised as a result of illness (Nieman & Pedersen, 2000). Acute endurance exercise ($>75\% \text{VO}_{2\text{max}}$) lasting for 1 hr or more has been reported to alter the immune system by depressing functional responses and changing the number of circulating leukocytes (Miles et al., 1998; Pedersen, Rohde, & Ostrowski, 1998). Less is known, however, about immune responses to resistance training. Studies examining immune responses to resistance exercise have consistently reported a significant increase in leukocytosis (Koch, Potteiger, Chan, Benedict, & Frey, 2001; Kraemer et al., 1996; Miles et al.; Nieman, Henson,

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et al., 1995; Shinkai, Shore, Shek, & Shepard, 1992). Nieman, Henson, et al. (1995) reported that a single session of resistance training induced leukocytosis and lymphocytosis immediately postexercise and lymphocytopenia 2 hr later. The degree of these immune responses was similar to those reported during and after high-intensity prolonged endurance exercise (Nehlsen-Cannarella et al., 1997).

The variations in leukocyte concentration during and after both endurance and resistance exercise might be the result of a temporary immunoredistribution between the peripheral lymphoid tissues and the circulation (Nieman, 1997; Shinkai et al., 1992). The alterations in differential leukocyte counts and function have also been linked, however, to exercise-induced changes in plasma cortisol concentrations (Koch et al., 2001; Nieman et al., 1995). During acute exercise, stress hormones such as cortisol have been shown to increase circulating leukocyte concentrations up to fourfold over basal values (Mackinnon, 2000). Conversely, elevations in cortisol are thought to lead to reductions in circulating lymphocytes during postexercise recovery (Shinkai, Watanbe, Asai, & Shek, 1996). Aside from exercise intensity, one of the major stimuli for the activation of the hypothalamic-pituitary-adrenal axis and the subsequent release of adrenocorticotropic hormone and cortisol is a reduction in circulating blood glucose (Nieman, 1998).

Recently, Koch et al. (2001) studied the effect of resistance exercise on immune function to determine whether carbohydrate supplementation would alter immune responses. They found that immediately after resistance exercise, leukocytosis, lymphocytosis, monocytosis, and neutrophilia were significantly elevated. In addition, there were no differences in postexercise immune responses in participants who consumed a carbohydrate supplement or placebo. Furthermore, plasma glucose and cortisol levels were equally elevated after resistance exercise with both carbohydrate and placebo treatments. Carbohydrate ingestion has been shown to attenuate cortisol responses with exercise of 75–80% $\text{VO}_{2\text{max}}$ and 2.5 hr of running (Henson et al., 1998; Nieman, Simandle, et al., 1995). Haff et al. (2000) found that an acute bout of resistance exercise lasting approximately 39 min decreased muscle glycogen content of the vastus lateralis by 26.7% compared with carbohydrate (CHO) supplementation (13.7% decline). It is possible that the duration and intensity of the resistance exercise in the study by Koch et al. were not sufficient to induce changes in plasma glucose concentrations such that carbohydrate supplementation would alter plasma cortisol responses and subsequent immune responses.

Given previous findings (Henson et al., 1998; Koch et al., 2001; Nieman, Simandle, et al., 1995), the aim of our investigation was to study changes in leukocyte subsets after an acute session of resistance exercise and to further determine whether the ingestion of CHO would attenuate those immune responses. We hypothesized that ingestion of a CHO beverage would attenuate the cortisol response and prevent subsequent lymphocyte reductions after resistance exercise.

Methods

Participants

This study consisted of two experiments, wherein moderately trained male college athletes (Experiment 1, $n = 9$; Experiment 2, $n = 10$) gave their written informed consent to participate in this study, which was approved by the university's

institutional review board, and completed a medical-history questionnaire. All participants were members of NCAA Division III athletic teams and had been training for at least 5 years, with a minimum of 2 years of weight-lifting experience. Participant characteristics for both experiments are presented in Table 1.

To minimize influence on the immune system, participants in both experiments adhered to instructions before attending exercise testing to not ingest caffeine, alcohol, or anti-inflammatory medications 24 hr before testing. In addition, participants agreed to abstain for 30 days from using large doses of vitamin/mineral supplements (>100% of recommended dietary allowances) until after the third exercise session. Participants were instructed not to engage in exercise during the 24 hr before each testing session.

Participants were excluded from the study if they had any immunocompromised condition such as an autoimmune disease (i.e., lupus, multiple sclerosis, rheumatoid arthritis, or insulin-dependent diabetes mellitus), tested positive for human immunodeficiency virus (HIV), or had been diagnosed with acquired immune deficiency syndrome (AIDS). Participants were also excluded if they were taking prescription medications, using steroids, using ergogenic supplements (e.g., creatine) for at least 1 month before testing or had indicated that they experienced high psychological stress. Before each testing session, participants who displayed any symptoms associated with URTI illness that would alter immune-cell parameters were excluded from the study.

Procedures

Dietary Control. During both experiments, participants were required to adhere to a macronutrient diet that consisted of the following percentages of their total energy intake: 40% CHO, 30% fat, and 30% protein. An example of the macronutrient meal plan was provided to the participants at the first session. For 2 days before the testing sessions, participants recorded their food intake. Dietary analyses were used to verify that the composition and energy content were similar for the 2 days before the testing sessions. Diet analyses were calculated using the Nutrition III diet-analysis software by N-Square Computing (Salem, OR).

Table 1 Participant Characteristics, $M \pm SD$

Characteristic	Experiment 1 ($n = 9$)	Experiment 2 ($n = 10$)
Age (years)	21.1 \pm 1.4	21.0 \pm 2.2
Height (cm)	177.2 \pm 5.5	174.3 \pm 6.2
Body weight (kg)	80.9 \pm 9.7	79.6 \pm 11.1
Body fat (%)	8.7 \pm 3.8	13.9 \pm 3.7
1-RM leg press (kg)	387.8 \pm 103.8	313.2 \pm 66.9
1-RM bench press (kg)	97.7 \pm 22.9	94.8 \pm 14.5
10-RM leg curl (kg)	38.5 \pm 10.6	53.4 \pm 11.0
10-RM lat pull-down (kg)	56.4 \pm 12.9	69.3 \pm 8.6
Years of training	5.0 \pm 2.4	4.5 \pm 1.5

Strength Assessment. One week before testing in both experiments, measurements of baseline height, body weight, and body composition via Jackson and Pollock's (1985) skinfold method were conducted. One-repetition maximums (1-RMs) using the 1-RM testing protocol (Bachle & Earle, 2000) were determined for the leg press (Cybex International, Medway, MA) and bench press (Sorinex Exercise Equipment, Irmo, SC), and 10-RMs were determined for the latissimus dorsi pull-down (York, PA) and leg curl (Cybex). The protocol for the 10-RM test was similar to the 1-RM, but each set required 10 repetitions.

Resistance-Exercise Protocol. In both experiments, on the second and third testing occasions participants were required to complete an exercise-session checklist before participation to confirm adherence to pretesting instructions. The resistance-exercise timeline used for both experiments is depicted graphically in Figure 1. Only the percent of 1-RM used in the warm-up phase of testing, the percent of 1-RM used for the actual lifting session, and the cadence of repetitions during the lifting session were different between Experiments 1 and 2. In both experiments, participants consumed either a CHO or P beverage before, during, or after the weight-lifting session. A randomized (on first day only) double-blind treatment condition was used with the exercise protocol. PowerAde was the beverage used (8% CHO solution). The CHO and P beverages were designed to be identical in appearance and taste, with the CHO concentration being the only difference. The P beverage contained aspartame, citric acid, food coloring, and acesulfame potassium (a high-intensity sweetener to make the product more palatable).

On both P and CHO days of testing, participants reported to the weight room after 12 hr of fasting. The temperature in the weight room was 22 °C for all testing sessions, and testing occurred at the same time of day for all participants. Participants were instructed to rest quietly in a seated position for 10 min before the first blood draw from an antecubital vein. After the first blood sample was collected, they consumed one third of a volume of fluid that contained 1 g of CHO per kilogram of body weight or an equal volume of P. After the beverage consumption and before the resistance exercise, participants stretched. Both the testing protocol and the 10-min time period after the beverage consumption were intended to prevent reactive hypoglycemia (Henson et al., 1998).

The resistance-exercise protocol in both experiments was designed to recruit and activate a large amount of muscle tissue by having participants perform exercises that use the major muscle groups in both the upper and the lower extremities. The exercise session consisted of two paired-exercise sets. The first paired-exercise set consisted of six sets of the leg press and six sets of latissimus dorsi pull-downs. The second set consisted of six sets of bench press and six sets of leg curls. In Experiment 1, the leg- and bench-press protocols consisted of two warm-up sets of 10 repetitions of that exercise at 35% and 45% of 1-RM and four sets of 10 repetitions at 55% of 1-RM. The lat-pull-down and leg-curl exercises also consisted of two warm-up sets of 10 repetitions of that exercise at 35% and 45% of 1-RM and four sets of 10 repetitions at 55% of 1-RM. The exercises were performed with a 3:2 cadence, and rest periods of 1 min were used between sets of exercises. The total time to complete the experiment 1 protocol was approximately 52 min. In Experiment 2, the leg- and bench-press protocols consisted of two warm-up sets of 10 repetitions of that exercise at 45% and 55% of 1-RM and four sets of 10 repetitions

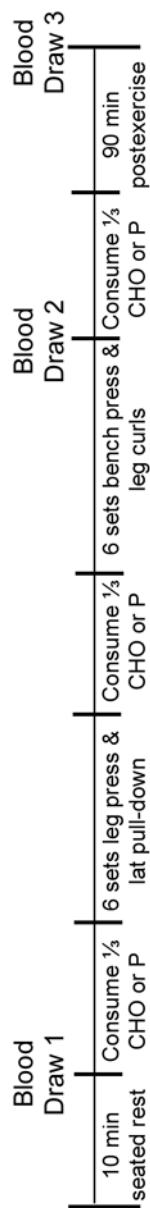


Figure 1 — Resistance-exercise protocol timeline. CHO = carbohydrate; P = placebo.

at 65% of 1-RM. The lat-pull-down and leg-curl exercises also consisted of two warm-up sets of 10 repetitions of that exercise at 45% and 55% of 1-RM and four sets of 10 repetitions at 65% of 1-RM. The exercises were performed with a 2:2 cadence, and rest periods of 1 min were used between sets of exercises. The total time to complete the Experiment 2 protocol was approximately 42 min.

After the completion of the exercise session in both experiments, a second blood sample was collected. A second dose of the same beverage and volume was administered immediately after the first paired-exercise set. A third dose of the same beverage and volume was provided after the second blood draw. At the completion of the lifting session, participants rested quietly for 90 min. The third blood sample was collected at the 90-min recovery point.

Blood Collection and Analyses. During Experiments 1 and 2, all three blood samples were drawn with the participants in a seated position. Blood samples were drawn at baseline, immediately postexercise, and after 90 min of recovery. Vacutainers containing anticoagulant (EDTA) were used for routine complete blood counts with automated differentials, and Vacutainers without additive (dry) were used for serum cortisol and glucose levels. Vacutainers containing sodium fluoride potassium oxalate were used for plasma lactate levels.

Blood samples containing the EDTA were sent for complete blood counts and leukocyte subsets via an automated differential to the Baystate Reference Laboratory (Springfield, MA). Total leukocytes and leukocyte subsets were corrected for changes in plasma volume via hematocrit and hemoglobin changes using the Dill and Costill (1974) method.

The blood samples for glucose, cortisol, and lactate were centrifuged for 10 min at 3,200 rpm after the blood draw, and the resulting serum and plasma was frozen at -40°C . Serum cortisol was assayed in triplicate using a competitive solid-phase ^{125}I radioimmunoassay technique (Biohealth Diagnostics, Santa Monica, CA). Serum glucose was assayed in duplicate via spectrophotometry using the hexokinase enzymatic method (Biohealth Diagnostics, Santa Monica, CA). Plasma lactate was assayed in duplicate via spectrophotometry (Sigma Kit #735, St. Louis, MO).

Statistical Analyses. For Experiments 1 and 2, a 2×3 (treatment by time) repeated-measures ANOVA for multiple comparisons was used to determine whether there were significant changes in the dependent variables within a treatment or between treatments. A Newman-Keuls post hoc analysis was used to isolate differences among treatment means. Previous studies of endurance athletes (Henson et al., 1998; Nieman, Simandle, et al., 1995) have reported attenuation of immune responses of up to 25–50% with CHO supplementation. Based on this observation, we assumed that a similar change could be expected in the current study and would be considered meaningful. From Vu Tran (1997), we estimated that 6–12 participants would provide sufficient statistical power ($\beta = 0.20$) and an alpha of .05 to detect a difference in immune responses.

Results

In the 2-day diet analysis before each time trial, no differences ($p > .05$) were found for kJ/day, percent CHO, percent fat, or percent protein consumed. The group averages for all trials were $10,571.62 \pm 4,384.63$ kJ/day, $43.5\% \pm 6.2\%$, 20.9%

$\pm 4.4\%$, and $35.9\% \pm 5.8\%$ for CHO, protein, and fat, respectively. Total volume (weight \cdot sets¹ \cdot reps¹) completed during the CHO and P exercise sessions was also not different and averaged $110,316 \pm 25,308$ kg.

For Experiment 2, again, no differences ($p > .05$) were found for kJ/day, percent CHO, percent fat, or percent protein consumed. The group averages for all trials were $10,088.4 \pm 2,268.4$ kJ/day, $45.6\% \pm 7.8\%$, $25.1\% \pm 3.2\%$, and $29.3\% \pm 5.2\%$ for CHO, protein, and fat, respectively. Total volume (weight \cdot sets¹ \cdot reps¹) completed during the CHO and P exercise sessions was also not different and averaged $118,239.6 \pm 19,199$ kg.

Immune Responses

In Experiment 1 (Table 2), a significant main effect ($p < .05$) for time was observed for leukocytes, lymphocytes, neutrophils, and monocytes. Post hoc analysis revealed that leukocytes, neutrophils, and monocytes were elevated immediately postexercise ($p < .05$) over preexercise values. At 90 min postexercise neutrophils remained elevated ($p < .05$), whereas lymphocytes were lower ($p < .05$) than preexercise values.

A treatment-by-time main effect ($p < .05$) was observed for lymphocytes. Post hoc analysis revealed a lower ($p < .05$) lymphocyte concentration in the CHO versus P treatment immediately postexercise. There were no time-by-treatment interactions ($p > .05$) observed between CHO and P treatments for differential leukocytes, neutrophils, or monocytes.

In Experiment 2 (Table 3), there was a significant main effect ($p < .05$) for time for leukocytes, lymphocytes, and neutrophils. Post hoc analysis revealed that immediately postexercise in the P treatment, lymphocytes were elevated ($p < .05$) above preexercise values. Immediately postexercise lymphocyte values in the CHO treatment remained unchanged ($p > .05$) from preexercise. Ninety minutes postexercise, leukocytes and neutrophils were elevated ($p < .05$) above preexercise and immediately postexercise values. Ninety-minute-postexercise lymphocyte values were lower ($p < .05$) than pre- and postexercise concentrations. There were no

Table 2 Experiment 1 Differential Leukocyte Responses ($10^9/L$) After Acute Resistance Exercise With Carbohydrate and Placebo Ingestion ($n = 9$), $M \pm SD$

Variable	Treatment	Preexercise	Postexercise	90 min postexercise
Leukocytes	carbohydrate	6.50 ± 1.99	$8.26 \pm 3.14^*$	7.14 ± 3.40
	placebo	5.91 ± 1.38	$8.10 \pm 3.24^*$	8.15 ± 3.71
Lymphocytes	carbohydrate	1.96 ± 1.38	$2.32 \pm 1.07^\dagger$	$1.26 \pm 0.41^*$
	placebo	2.02 ± 0.48	2.98 ± 1.43	$1.50 \pm 0.54^*$
Neutrophils	carbohydrate	3.59 ± 1.49	$4.59 \pm 2.26^*$	$5.74 \pm 3.75^*$
	placebo	2.98 ± 1.01	$3.97 \pm 1.72^*$	$6.79 \pm 3.82^*$
Monocytes	carbohydrate	0.74 ± 0.23	$0.96 \pm 0.37^*$	0.76 ± 0.27
	placebo	0.66 ± 0.19	$0.88 \pm 0.38^*$	0.66 ± 0.25

*Significant difference from preexercise value within a treatment ($p < .05$).

† Significant difference between treatments at corresponding time point ($p < .05$).

Table 3 Experiment 2 Differential Leukocyte Responses ($10^9/L$) After Acute Resistance Exercise With Carbohydrate and Placebo Ingestion ($n = 10$), $M \pm SD$

Variable	Treatment	Preexercise	Postexercise	90 min postexercise
Leukocytes	carbohydrate	6.42 ± 1.20	7.61 ± 2.02	$7.40 \pm 3.20^*$
	placebo	6.67 ± 1.70	8.24 ± 2.03	8.8 ± 3.50
Lymphocytes	carbohydrate	2.54 ± 0.57	2.60 ± 0.64	$1.68 \pm 0.44^{*\ddagger}$
	placebo	2.74 ± 0.82	$3.23 \pm 0.92^*$	$1.57 \pm 0.49^{*\ddagger}$
Neutrophils	carbohydrate	3.20 ± 0.91	4.11 ± 1.76	$5.01 \pm 3.18^*$
	placebo	3.20 ± 0.70	4.00 ± 1.20	$6.40 \pm 3.31^{*\ddagger}$

*Significant difference from preexercise value within a treatment ($p < .05$).

\ddagger Significant difference from postexercise value within a treatment ($p < .05$).

time-by-treatment interactions ($p > .05$) observed between CHO and P treatments for differential leukocytes, lymphocytes, or neutrophils.

Cortisol, Glucose, and Lactate Responses

During Experiment 1 (Table 4), there was a significant main effect for time ($p < .05$) observed for serum cortisol, serum glucose, and plasma lactate. Serum glucose and plasma lactate values were greater immediately postexercise ($p < .05$) than preexercise. At 90 min postexercise, plasma lactate values were lower ($p < .05$) than postexercise. At 90 min postexercise serum cortisol values were lower ($p < .05$) than preexercise values. There were no time-by-treatment interactions ($p > .05$) observed between CHO and P treatments for serum cortisol, serum glucose, or plasma lactate concentrations.

During Experiment 2 (Table 5), there was a significant main effect for time ($p < .05$) observed for serum glucose and plasma lactate. Immediately postexercise plasma lactate values were elevated ($p < .05$) above preexercise values. By 90 min postexercise, plasma lactate values were lower ($p < .05$) than immediately postexercise but were greater ($p < .05$) than they had been preexercise. By 90 min postexercise in the CHO treatment, serum glucose values were lower than pre- and immediately postexercise concentrations.

Discussion

Suppression of lymphocyte count and proliferation has been reported after prolonged endurance and resistance exercise (Nehlsen-Cannarella et al., 1997; Nieman, Henson, et al., 1995; Nieman, 1997; Nieman, 2000). The current investigation sought to examine CHO supplementation on cortisol and subsequent leukocyte responses after acute resistive exercise. Our primary finding was that although the resistance exercise stimulated leukocyte mobilization, the ingestion of CHO had no effect on serum cortisol, nor did it alter leukocyte, neutrophil, or monocyte concentrations. CHO supplementation might, however, have attenuated the lymphocyte count immediately postexercise.

Table 4 Experiment 1 Cortisol, Glucose, and Lactate Responses (mmol/L) After Acute Resistance Exercise With Carbohydrate and Placebo Ingestion ($n = 9$), $M \pm SD$

Variable	Treatment	Preexercise	Postexercise	90 min postexercise
Cortisol	carbohydrate	452 \pm 65	473 \pm 171	328 \pm 120*
	placebo	447 \pm 78	485 \pm 121	408 \pm 146*
Glucose	carbohydrate	5.4 \pm 0.4	6.0 \pm 1.2*	4.4 \pm 0.6
	placebo	5.0 \pm 0.9	6.0 \pm 1.3*	5.2 \pm 0.4
Lactate	carbohydrate	1.43 \pm 0.16	13.67 \pm 0.92*	2.48 \pm 0.38†
	placebo	1.38 \pm 0.11	13.38 \pm 1.01*	2.41 \pm 0.36†

*Significant difference from preexercise value within a treatment ($p < .05$).

†Significant difference ■

Table 5 Experiment 2 Cortisol, Glucose, and Lactate Responses (mmol/L) After Acute Resistance Exercise With Carbohydrate and Placebo Ingestion ($n = 10$), $M \pm SD$

Variable	Treatment	Preexercise	Postexercise	90 min postexercise
Cortisol	carbohydrate	712 \pm 127	644 \pm 171	540 \pm 197
	placebo	684 \pm 121	847 \pm 313	703 \pm 187
Glucose	carbohydrate	5.4 \pm 0.4	5.8 \pm 1.4	3.9 \pm 0.8*‡
	placebo	5.2 \pm 0.3	5.7 \pm 1.9	4.7 \pm 0.9
Lactate	carbohydrate	2.36 \pm 0.75	13.60 \pm 2.20*	4.80 \pm 1.20*‡
	placebo	3.23 \pm 3.0	14.47 \pm 2.45*	6.02 \pm 3.70*‡

*Significant difference from preexercise value within a treatment ($p < .05$).

‡Significant difference from postexercise value within a treatment ($p < .05$).

Immune Responses

Acute bouts of heavy endurance or resistance exercise have been shown to influence immune cells in the circulating peripheral blood (Henson et al., 1999, 1998; Kraemer et al., 1996; Nieman, 1997; Nieman, Henson, et al., 1995; Nieman, Simandle, et al., 1995; Simonson & Jackson, 2004■). Concurrent with several reports (Kraemer et al.; Mitchell et al., 1998; Nieman, Henson, et al., 1995; Nieman, Simandle, et al., 1995; Simonson & Jackson, 2004), we observed increases in total leukocyte, lymphocytes, and neutrophils immediately after acute resistive exercise. We did not, however, observe the same magnitude of cell responses with resistive exercise as that reported during endurance exercise. It is possible that the magnitude of exercise intensity and the duration of the resistance exercise in the current study were appreciably shorter and less intense than during the previous endurance-exercise protocols.

Our findings, in accordance with previous literature, demonstrate that an acute bout of resistance exercise influences circulating leukocytes and their subsets postexercise. CHO ingestion has been associated with an attenuated immunosuppression

by blunting the cortisol response during prolonged endurance exercise (Nieman, 1998; Nieman et al., 2001). In the current study, the CHO beverage was effective in attenuating lymphocyte numbers postexercise. The immediately postexercise increase in lymphocytes is likely caused by an increase in natural killer (NK) cells (Pedersen et al., 1997). It is possible that a greater rise in NK cells occurred in the P treatment because of the lack of supplemental CHO (Figure 2). During the P trial the lower carbohydrate availability during the resistance protocol could have represented a greater physiological challenge, thus stimulating a greater catecholamine response and NK-cell increase (Pedersen et al., 1997). This response would likely not reflect a suppression of immunity with the CHO treatment but, rather, a decrease in physiological stress as a result of increased CHO availability. By 90 min postexercise, however, there was no significant difference in lymphocyte

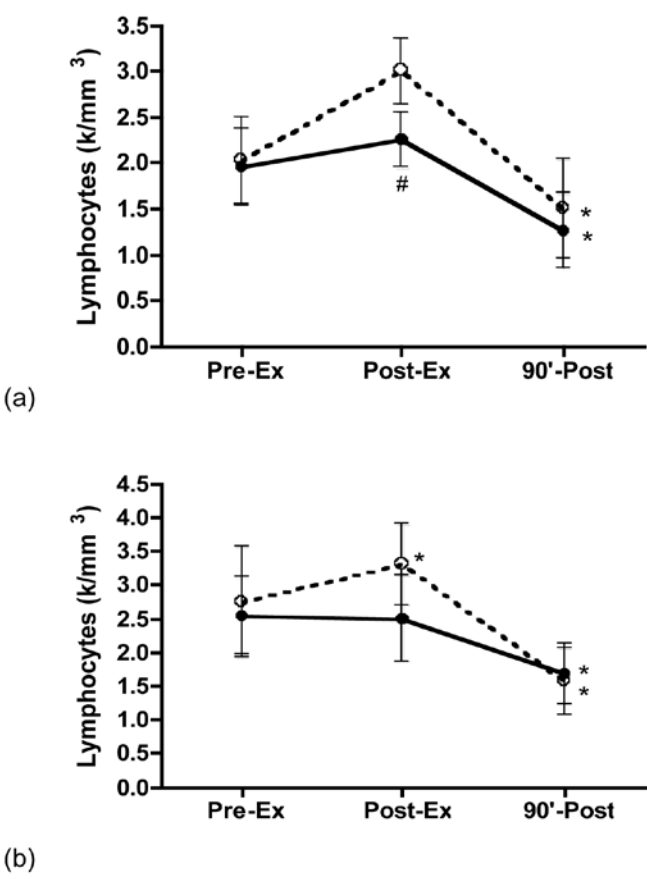


Figure 2 — Lymphocyte responses ($M \pm SD$) preexercise, postexercise, and after 90-min recovery for carbohydrate (bcirc) and placebo (wcirc) treatments for (a) Experiment 1 and (b) Experiment 2. #Significant difference between treatments at corresponding time point ($p < .05$). *Significant difference from baseline value within a treatment ($p < .05$).

concentrations between the treatments, because the resistance-exercise challenge was no longer present.

Lymphocytes are typically activated when there is an immune challenge such as exposure to antigens (Nieman, 1999, 2000). According to Nieman, Henson, et al. (1995), resistance exercise causes an increase in epinephrine, which increases the sensitivity of lymphocytic surface receptors. As a result, the lymphocytes lose their adhesion to the endothelium and migrate into circulation, producing an exercise-induced lymphocytosis observed immediately postexercise (Nieman, Henson, et al., 1995). Völker et al. (2007) reported that intensive resistance exercise significantly increased leukocyte counts postexercise, and the lymphocytes declined 3 hr into recovery. Further analysis confirmed that the postexercise decline in lymphocyte count was attributable to increases of apoptotic lymphocytes. CHO supplementation might decrease the physiological strain by increasing CHO availability, thus lessening a sympathetic response, catecholamine release, and subsequent variation in lymphocyte count. Whether this response is physiologically beneficial should be further investigated.

Cortisol Response With CHO Supplementation

Elevations in cortisol, in response to resistance training, occur when the rest period is brief (1 min; Mitchell et al., 1998). Kraemer et al. (1996) suggested that the duration of the force production and rest period are the key variables that influence the cortisol response to resistance exercise. They reported, however, that leg-press exercises with both 1- and 3-min rest periods between sets elicited leukocytosis without significant elevations in cortisol. Koch et al. (2001) found significant elevations in cortisol from baseline to postexercise but no differences with regard to CHO supplementation. As a result, Koch et al. suggested that the exercise protocol be longer in duration.

The current study attempted to increase the duration of the exercise sessions from 15 min to 52 min and 42 min; however, this did not elicit a significant increase or difference in serum cortisol levels between the treatments. One reason for lack of a significant increase in cortisol might be the intensity of the resistance exercises. An attempt was made to prolong the duration of the resistance-exercise protocol; however, as a result, the intensity or force production was reduced. Another possible reason for the decline in serum cortisol postexercise might be normal diurnal variation; we started testing in the morning when cortisol was at its peak.

Conclusions

The acute bout of resistance exercise used in this study did result in an increase in immune responses, although not to the same degree as that reported with endurance exercise. In addition, the CHO treatment did not alter serum concentrations of cortisol or responses of leukocytes, neutrophils, or monocytes. It is possible that the intensity and duration of the resistance-exercise protocol were not great enough to elicit a significant increase in cortisol responses. CHO supplementation was associated with lower lymphocyte counts immediately postexercise, but at 90-min recovery after exercise lymphocyte production was not different between the CHO supplementation and P treatment.

Applications

Although CHO supplementation might not directly attenuate immune responses as a result of suppressing the release of cortisol, the greater CHO availability might decrease the physiological strain by increasing CHO availability, thus lessening a sympathetic response, catecholamine release, and lymphocyte mobility. If this is the case, CHO supplementation either during or immediately after resistance exercise might be beneficial in decreasing physiological strain and lymphocyte migration to the peripheral blood. It is also possible that resistance exercise of greater duration or intensity could alter immune function such that CHO supplementation would be beneficial.

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